Chapter 20

ACUTE OTOLOGICAL TRAUMA

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INTRODUCTION

Otological injuries have always been a part of warfare, but modern combat seems to be particularly hard on the ear, hearing, and balance. This chapter and Chapter 36, Delayed Otological Trauma are based on first-hand knowledge of these facts. These chapters are meant to complement each other, but some overlap exists because the transition from one environment to another is not always so clear-cut. This is especially true when caring for the indigenous population because acute and definitive care are provided in theater.

Blasts have been the most common cause of ear trauma in Iraq and Afghanistan, but often in combination with other conditions. Each patient somehow suffers a unique injury. However, in general, otological injuries can be separated into five categories:

1. tympanic membrane perforation,
2. acute sensorineural hearing loss and tinnitus,
3. acute dizziness,
4. aural soft-tissue trauma, and
5. temporal bone trauma.

Each injury can occur in isolation or in combination. Blunt temporal bone fractures, facial nerve injuries, and vascular injuries are relevant subjects and are described elsewhere in this volume. Dizziness and sensorineural hearing loss are also examined, but they are specifically reviewed in detail here to emphasize acute management. Therefore, practical tips for otology in an austere environment will be germane, even if there is Role 4 evidence.

Each war brings its own lessons, whereas only some will apply to the next war. Thus, military otolaryngologists had a lot to learn following 9/11. There was almost no practical experience with combat otological trauma (especially from improvised explosive devices [IEDs]), and the literature seemed irrelevant or outdated. Otolaryngologists primarily learned on their own when they started treating wounded soldiers; but once they began collaborating with each other, things improved. Thus, there is a different back story for each topic in this chapter. This is especially true for posttraumatic dizziness, which must have always been a factor, but did not register any major attention until recently. The literature on dizziness that is significant came from events of the last decade, but there remain a lot of important unanswered questions.

What can be done for patients depends on overall medical status, what equipment and resources are available, and if (or when) they will be evacuated. It is not unusual to see a patient who was intubated in the field and who stays that way until theater evacuation. The patient's available medical history gathered at the time of trauma is often cursory at best, and the otolaryngologist will be unable to ask about hearing loss, tinnitus, or dizziness. Otological injuries will often take a backseat even though they are relevant and common. Some assumptions are necessary, and it is prudent to examine the ears and respond appropriately just in case things are overlooked at the next level of care. It may be a while before an otolaryngologist is involved again.

Deployment medicine does not suit the rigid mind. A flexible, pragmatic approach is necessary to be effective. Many problems—especially with severe trauma—only have partial solutions, or the provider may even devise a good solution that eventually ends up foiled by some other factor. This becomes quickly frustrating, especially if higher level personnel second-guess the actions of the otolaryngologist. The best method involves keeping emotions in check, listening, and trying to understand the other point of view. On one hand, if the physician is satisfied with treatment and others would not have done anything differently, then it is necessary that the physician carries on with that particular treatment protocol. However, on the other hand, it is important to note that even the most experienced and prepared surgeons can be occasionally enlightened about better treatment options if they keep an open mind.

Ultimately, providers will need to make nuanced decisions about what to treat and what to leave for colleagues to treat later. Factors in these decisions will vary according to deployment location, logistics, and operation tempo. Every provider is part of the team, and each physician should choose which treatments and resources to use for the greatest overall good of patients. The convenience of any provider is secondary.

TYMPANIC MEMBRANE PERFORATION

During any initial trauma evaluation, it is essential to look for tympanic membrane perforations. They are common after blasts and were present in 15% of 436 explosion-wounded patients evacuated to Brooke Army Medical Center (Fort Sam Houston, TX); half of these cases required tympanoplasty.[1] Tympanic membrane perforations may also be caused by penetrating trauma, blunt skull base trauma, hand-to-hand combat, barotrauma, etc. These perforations are not something to definitively operate on in theater (at

least acutely), but some preliminary management can be done with a microscope. If a microscope is not available, then try using a headlight, loupes, and a small nasal speculum. The acute otological assessment and management are frequently performed in conjunction with other ongoing management. If a patient is going to the operating room for other reasons, then it is vital to get in and work with the rest of the operative team. However, it may be awkward, but it also may be the only chance to do something meaningful for the patient before the next stage of evacuation. A view of the tympanic membrane is often obscured with blood, which is easily washed out with antibiotic drops. Fragments, wood, dirt, and pebbles are also common, but require more effort. Remove as much debris as possible. Sterile irrigation can be used like in any other ear surgery, but be cautious if there is a skull base defect. Always attempt to unfurl what remains of the tympanic membrane. This action should improve healing and decrease the chance that these edges of epithelium will form a cholesteatoma. Much of this treatment is better performed under anesthesia (when available), especially for the occasional ear surgeon.

Use ototopical antibiotic drops whenever a perforation is dirty or wet. Fluoroquinolones are preferred, whereas anything with an aminoglycoside should be strictly avoided because of ototoxicity. Write the order for 1 week or 10 days at most because problems occur when patients have been on drops for several weeks or even months. This "Ciprodex overkill" (Ciprodex, Alcon Laboratories, Inc, Fort Worth, TX) does not add any benefit to a week of treatment, but it does encourage fungal superinfection. There is no need to use drops for a clean, dry perforation unless there is an acute sensorineural hearing loss (SNHL) or tinnitus. So, in that case, consider drops containing a steroid because of its otoprotective effect. It is possible that the steroid, in combination with antibiotic/steroid drops, will delay the healing of the tympanic membrane,[2,3] but this seems less of a problem than permanent hearing loss and tinnitus.

ACUTE SENSORINEURAL HEARING LOSS AND TINNITUS

Combat causes a lot of hearing loss and tinnitus. According to the US Department of Veterans Affairs, these continue to be the two most prevalent service-related disabilities.[4] In 2010, 63,583 veterans with hearing loss and 92,260 veterans with tinnitus began receiving compensation. These cases were primarily from the Global War on Terrorism, and this was a 15% increase from the prior year.[4] This staggering problem is multifaceted, but we have a responsibility to do something about it postexposure. Unfortunately, hearing loss and tinnitus are frequently overlooked or ignored at first. They may not seem important when a patient has other injuries, but this is a shortsighted view because immediate treatments may have lasting results. Inner ear injury is frequently permanent, but there is a window of opportunity for many, and this should not be squandered.

The potential for long-term hearing loss depends on the type and level of noise exposure. Impulse noise is more damaging than continuous sound, and the mechanism of cochlear damage shifts from biochemical to mechanical injury as the sound energy increases.[5] The incidence of SNHL after a blast-induced tympanic membrane perforation was 36% for those requiring tympanoplasty at Walter Reed National Military Medical Center (Bethesda, MD) and when present averaged 60 dB at 6 or 8 kHz.[6] Other series have put this incidence as high as 78%,[7] but it is hard to compare vastly different groups. Hearing loss from blasts tends to stabilize to higher frequencies.[1,6] It often looks like the "noise notch" seen with chronic noise exposure, but the spectrum of SNHL from acoustic trauma is broad. An IED blast triple amputee even required a cochlear implant while he was at Walter Reed, but this was extremely unusual. About 5% of those who require tympanoplasty eventually are fitted with a hearing aid,[1] and tinnitus is a serious problem for many whatever the magnitude of hearing loss.[8–10]

Providers, the command, and patients can easily discount acute hearing loss if there are no visible wounds. Also, there does not have to be blood draining from the ears to indicate an acute threshold shift. An otherwise well patient may present with disabling hearing loss and/or tinnitus after a specific incident (eg, firing a weapon without proper hearing protection). This needs to be taken seriously. The examination should at least include a Weber test. Lateralization to the uninjured ear would be a worrisome finding that should be documented, but this will only be specific for low-frequency hearing losses. An audiogram is ideal to quantify and follow the magnitude of the hearing loss, but it is not always reasonable (or possible) to obtain one in theater. For example, there were no audiologists in Afghanistan during one author's deployment (MJR), but the other author (PDL) intermittently had access to an audiologist in Iraq. However, an interesting question to ask is, "Would an audiogram change patient treatment?" It probably would not unless the results are completely normal, and if the patient does not have tinnitus. It is better to presume an acute SNHL and

immediately start treatment when a patient complains of symptoms following noise exposure than to expend resources (and potentially risk lives) for transport to an audiologist. Objective verification with an audiogram is not necessary for treatment in this situation.

There is a growing body of evidence that shows that several compounds can decrease hearing loss if used soon after noise trauma. This is an active area of research; however, it is difficult to give any evidence-based recommendations from the available literature. The information below is a brief review of the current literature, and Exhibit 20-1 summarizes our guidelines based on available evidence (as well as some intuition).

Steroids are by far the most common and familiar otoprotectives, although it is hard to say if they are the standard of care for acute noise-induced SNHL. They probably work through several mechanisms, including scavenging of oxygen free radicals and inhibition of lipid peroxidation.[11] Topical steroids have shown a strong, acute otoprotective effect in studies of animals exposed to impulse noise[12,13] or drill trauma.[14] However, one study on guinea pigs after gunshot noise showed that while topical steroids accelerated hearing recovery and decreased hair cell loss, they did not make a difference on the long-term thresholds.[15] There are no controlled clinical trials evaluating systemic steroids after noise trauma, but there is a recent one for intratympanic (IT) steroids.

In a randomized controlled trial by Zhou et al,[16] 53 consecutive patients with hearing loss from noise trauma were hospitalized (in China) and treated with 10 days of parenteral methylprednisolone, naftidrofuryl, and low-molecular-weight heparin or low-molecular-weight dextran. Of these, 27 patients were also treated with IT methylprednisolone every other day. To be included in the study, there had to be at least one definite episode of intense noise exposure within the prior 2 weeks, and a SNHL with an average of ≥40 dB at 2, 4, and 6 kHz. Furthermore, this hearing loss was monitored for 3 days, and patients were excluded if spontaneous recovery of >15 dB occurred. At 8 weeks follow-up, 51.9% of the treatment group had an improvement of ≥15 dB versus 23.1% of the control group. For speech discrimination, 66.7% of the treatment group had ≥15% improvement versus 30.8% in the control group. All of these results were statistically significant. This study suggests that IT steroids improve hearing in patients with recent noise trauma. This benefit was in addition to any benefit of parenteral steroids, vasodilators, and plasma expanders provided in the control group.

Other compounds with more specific mechanisms have also been effective in laboratory animals exposed to impulse noise. These include the following:

EXHIBIT 20-1

TREATMENT GUIDELINES FOR ACUTE BLAST/ NOISE-INDUCED SENSORINEURAL HEARING LOSS

Who?
- Patients with intense combat blast/noise exposure and suspected SNHL or tinnitus
- The worse the injury and symptoms, the more important the treatment

When?
- As soon as possible
- Within 10 days of injury, possibly longer for severe injuries

How?
- Noise rest
- *N*-acetylcysteine: oral or IV (consider a loading dose)
- *and/or*
- Steroids: oral or IV (consider a loading dose) *also consider adding*
- Intratympanic steroids, if the SNHL seems severe
- Topical steroids, if there is a tympanic membrane perforation

How Long?
- Around 14 days

IV: intravenous; SNHL: sensorineural hearing loss

- antioxidants such as acetyl-L-carnitine and *N*-acetylcysteine (NAC)[17–19];
- compounds that decrease reactive nitrogen species, such as astragaloside IV[20];
- leupeptin, a calpain inhibitor (a family of calcium-activated proteases that promote cell death)[21];
- Src-protein kinase inhibitors (part of the apoptotic signaling mechanism)[22];
- AM-111 (an inhibitor of JNK-mediated apoptosis)[23]; and
- others.

Caroverine and magnesium are also effective in laboratory animals, but their exact mechanisms are unknown.[24,25] Outcomes in studies utilizing NAC after continuous broadband noise exposure have produced negative results, thus tempering enthusiasm.[26,27] In addition to impulse noise experiments, rats have also been tested after injury in a blast simulator. The combination of antioxidants 2,4-disulfonyl α-phenyl tertiary butyl nitrone (or HPN-07) and NAC caused a significant difference in auditory-evoked brainstem response threshold

shifts, and decreased outer hair cell loss (from 32% in the control group to 10% in the treatment group) at 21 days postexposure.[28]

These results have translated into several encouraging clinical studies. NAC was tested on military officers immediately after indoor firearms training, and it had a small otoprotective effect.[29] In a prospective, double-blind crossover study on Chinese workers, prophylactic NAC had a significant preventative effect reducing temporary threshold shifts after their workday.[30] It did not appear to help after loud music.[31] Although all of this is interesting, it does not do any good if these compounds are impractical or too novel to be available. NAC is already available in military pharmacies because it is the antidote for acetaminophen overdose and is also nebulized for respiratory therapy. It was used for a traumatic brain injury (TBI) study in Iraq, and hearing loss was one symptom that was followed.[32] NAC was given as a 4 to 8 g loading dose (tablet), followed with 2 g bid for 7 days. It was only available to providers in intravenous or inhaled formulations, but tablets are available over the counter as a dietary supplement. Tablets are definitely the more palatable NAC formulation because they do not have a sulfurous odor. Another advantage is that oral preparations are inexpensive.

The ideal timing of pharmacotherapy is not known, but sooner appears to be the better option. Questions arise when a patient presents several days postexposure, for example: How late is too late? When do the risks and expenses of therapy outweigh the possibility of benefit? In the study by Zhou et al,[16] the treatment group started IT steroids 5.6 ± 3.4 days postexposure and had hearing improvements greater than in a similar control group (4.8 ± 2.8 days). Animal data may also give some insight. One study showed that NAC and other antioxidants still had an otoprotective effect if given 10 days postimpulse noise injury.[33] Meanwhile, another animal study showed that methylprednisolone only had a protective effect if applied before or immediately after trauma, but not after 3 hours.[34] This topic requires further investigation, but it has been found that 10 days (or longer) is a fair estimate if hearing loss is profound.

Hearing loss can be prevented if there is a reasonable chance. Thus, a low threshold for otoprotective treatment is recommended after any noise trauma. Practically speaking, this will be a steroid and/or NAC. Empirical treatment seems justified because there is evidence supporting its use, there is a low risk-to-benefit ratio, and there is the fact that definitive studies are very challenging and probably far off in the future. Steroids do have their risks, but they are small compared with the potential benefits of hearing. Counsel accordingly about their side effects and rare, but serious, risks. If more specific compounds were immediately available at the unit level, treatment could be improved. However, it is not yet a reality.

What happens if a patient is unconscious following blast trauma? There is a good chance that the patient has an SNHL, especially if there is a tympanic membrane perforation. The problem is that the patient is unable to complain about it, and the provider is unable to evaluate it. It is reasonable to start pharmacotherapy, but this can become complicated. Any patient with an intracranial injury will probably be on parenteral steroids, but trauma and orthopaedic surgeons may be reluctant to use them when there are core or limb injuries. These concerns are best respected, but the issue is avoided by using topical steroids whenever there is a tympanic membrane perforation or by using NAC.

There is a lot of variation in steroid dosing among otologists, but a typical oral regimen is prednisone 60 mg once a day for 4 days, then decreasing it to 20-mg intervals for a total treatment period of 10 days. Other durations, doses, and steroids (ie, a methylprednisolone dose pack) have their advocates, but the key is to give something acutely. Even consider an intravenous loading dose if an intravenous line is already present.

In light of the preceding review, it seems reasonable to offer some type of steroid treatment for acute, noise-induced hearing loss. Systemic and/or IT dosing could be used. Exclusively, IT administration is appropriate if there are contraindications to systemic treatment. Local therapy has its advantages. For example, when compared with systemic steroids, the average drug concentration in the cochlea is at least twice as high with IT perfusion.[35] However, more research is needed to clarify the ideal routes and dosing of therapy. It is also important to define specific audiological criteria for any therapy because different severities of hearing loss will have different outcomes with and without treatment.

Opinions and techniques for IT delivery vary widely, but a tympanic membrane perforation simplifies it greatly. A combination antibiotic/steroid drop is appropriate if the perforation is dirty. The concentration of dexamethasone in ciprofloxacin/dexamethasone is 1 mg/mL, which is substantial, but less than typical dexamethasone solutions. The following procedure (based on a technique by Robert Battista[36]) works well if the tympanic membrane is intact:

- Apply topical phenol to the tympanic membrane with a swab and then inject 0.4 mL of dexamethasone (4 or 10 mg/mL) or methylprednisolone (125 mg/2 mL). The location of the injection probably does not matter as long as the solution accumulates in the oval and round window regions.

- Use a 1-mL syringe and inject with a 1.25-inch, 27-gauge needle (bend the needle by about 10 degrees at the hub). A microscope is ideal for this procedure, but it can also be done with a headlight and loupes or even through an otoscope; however, a longer needle is necessary (usually a 2.5-inch, 25-gauge spinal).
- Have the patient lay with the injected ear up for 30 minutes to allow absorption through the round window membrane.
- Repeat 2 to 3 times over a 2-week period. Any IT injection can cause brief caloric vertigo, and methylprednisolone usually causes a burning sensation. Be sure to warn the patient beforehand.
- Do not add a local anesthetic to the injection, which can cause hours of intense vertigo.

A patient with an acute hearing loss can usually return to duty while taking oral steroids, but there are exceptions. One simply may not be able to hear well enough to perform, especially with bilateral hearing loss. This should be explained to the patient and to command. Furthermore, some patients experience severe behavioral side effects from systemic steroids. This may require a brief change of duty, but it can be worked out within the unit. IT steroids will be a logistical problem for patients far from treatment centers, so it is probably best to keep them nearby for a couple of weeks. A recovery period of this length seems reasonable, especially since it will minimize hazardous noise exposure when their auditory hair cells are less able to tolerate it. Consider redeployment with hearing aids after a couple of weeks if the patient is still unable to hear well enough to perform.

It is possible that an acoustically rich environment immediately after noise trauma decreases the cortical frequency-place remapping that can lead to chronic tinnitus.[37,38] So far, this has only been studied in cats; but, if it is true, then patients should not be placed in perfectly quiet quarters after a noise trauma. As therapy, sound could be delivered around the frequencies of the hearing loss as long as it is not intense enough to exacerbate cellular damage. Although enticing, there are no studies in humans, and this appears to make chronic tinnitus worse.[39] For now, moderation is the best recommendation because of these contradictions.

A patient with an acute unilateral SNHL typically requires an MRI (magnetic resonance imaging) to rule out a retrocochlear lesion. This is not reasonable in theater and does not make much sense when the hearing loss is temporally related to a trauma. MRIs are not important for SNHL after otological trauma, but there are always exceptions that prevent strong opinions. One author (PDL) resected a vestibular schwannoma that was diagnosed by MRI shortly after a blast made the patient seek care for tinnitus. Still, if someone had normal hearing and now complains of hearing loss immediately after a specific trauma (eg, blast), and has no other neurological findings, then it is reasonable to assume that it is the result of the blast. There may be a predeployment hearing conservation audiogram for comparison. Regardless, any hearing loss should be followed up after return to the continental United States to ensure that it does not progress.

ACUTE DIZZINESS

Most patients with posttraumatic dizziness do not seem to present acutely. There are many possible reasons for this. Perhaps other injuries/issues take precedence. Sometimes symptoms do not manifest until patients are out of bed and moving about. It is also possible that the symptoms get worse as posttraumatic anxiety develops. More importantly, in-theater patients with balance disorders are frequently marginalized because dizziness is vague, the differential is extensive, and it is an invisible disorder. Patients with severe traumas and imbalance are evacuated. The remaining patients suffering with dizziness may look fine, but in reality have sustained significant injuries. These patients may need a lot of help to regain function.

There is no agreed-upon evaluation for dizziness, although the Department of Defense's Hearing Center of Excellence is working to standardize the process to improve patient care and data quality. Exhibit 20-2 outlines the basic bedside examination used by one author (PDL). It is the same procedure used when deployed as it is when nondeployed. Unfortunately, the examination has its limitations, and experts disagree on the usefulness of any given test. It is quite possible for a patient to be very dizzy (even have vertigo), yet still have an entirely normal examination. One reason for this is that voluntary movements and reflexes are tested, but not actual perception. However, findings like abnormal smooth pursuit or a positive Romberg test are highly variable and nonlocalizing. Some findings like spontaneous nystagmus and positioning nystagmus do localize pathology and guide care, but they are not always present.

There are also efforts to develop portable technologies that will make the examination for dizziness easier and more insightful, but for now the bedside examination is used. This needs to be completed and

EXHIBIT 20-2

BASIC BEDSIDE EXAMINATION FOR DIZZINESS

Standard Tests
Otoscopic examination
Cranial nerve assessment
Eyes
- Pupils and convergence
- Cardinal positions of gaze (also look for gaze-evoked nystagmus)
- Smooth pursuit
- Saccades

Nystagmus (with VNG goggles or Fresnel lenses, when available)
- Spontaneous and gaze-evoked nystagmus
- Neck rotation (evaluate for ease of rotation, symptoms, and nystagmus)
- Dix-Hallpike maneuvers
- Supine head hang
- Post headshake

Head thrust
Cerebellar testing
- Finger to nose
- Rapid alternating movements
- Pronator drift
- Heel to shin

Romberg
Gait

Optional Tests (Depending on History and Examination)
- Dynamic visual acuity
- Valsalva against closed glottis and then pinched nostrils
- Hyperventilation (does it induce nystagmus or reproduce symptoms?)
- Deep head hang and Lempert roll (for anterior and lateral canal BPPV)

BPPV: benign paroxysmal positional vertigo;
VNG: videonystagmography

documented so that the examination testing can be appropriately categorized according to importance. Providers know that removing visual fixation enhances vestibular nystagmus. This process is ideally performed wearing videonystagmography goggles, or at least Fresnel lenses, but these technologies are hard to come by. Thus, fixation can be decreased by having the patient stare at a blank sheet of paper during mental tasking (eg, counting backwards in sevens, naming states that begin with a certain letter, etc).

Aural or acoustic trauma may produce unilateral or asymmetric bilateral vestibular lesions. However, a combination of vertigo and oscillopsia with spontaneous nystagmus can occur or even an abnormal head thrust test. Oscillopsia is usually considered a sign of bilateral injury, but it is also common soon after a large unilateral lesion. These patients recover through vestibular compensation—like any other patient with an acute vestibular lesion—as long as there are not any medications, activity limitations, or accompanying central neurological injuries that interfere with the process. Patients should resume activities of daily living as soon as possible. Normal head and body motions will compensate the vestibuloocular and vestibulospinal reflexes, but some patients need activities that are more zealous to regain prior levels of function. Physical therapy aids compensation, but may not be practical in the deployed setting.

Head trauma patients most commonly complain of indistinct nonvertigo dizziness.[40] The provider will not get many details unless he/she asks the patient. Therefore, it is important for the physician to identify the primary symptom as vertigo, oscillopsia, dysequilibrium, imbalance, or lightheadedness. Generally, start treatment as if there is a peripheral lesion, knowing that more than one problem may have to be treated. Cervicogenic dizziness is typical of this and is seen following combat trauma. It is characterized by a sensation of wobbly disequilibrium. It usually resolves itself without treatment, but occasionally physical therapy exercises that emphasize cervical proprioception are beneficial.[41] These exercises are not difficult and are readily found online if a therapist is not available.

Dizziness is one of the most common symptoms of combat-related mild TBI (mTBI).[42] This is a complicated, controversial, and political topic, but some of these symptoms probably come from damage to central vestibular circuits.[43–45] These patients generally seem to recover from their dizziness in time, but not always. Physical therapy emphasizing vestibular rehabilitation can accelerate recovery, although it is important not to push too hard or too soon.[46] Acutely, it seems best to allow a patient some time to rest, and then gradually increase activity. TBI can cause autonomic dysfunction and lightheadedness with standing,[46,47] but so does inactivity. Thus, it is important to eventually get these patients up and moving about.

Neuroprotectives might ameliorate the symptoms of TBI (including dizziness) if given acutely. In a randomized, double-blind, placebo-controlled trial in Iraq, NAC appeared to do just this when given less than 72 hours after mTBI.[32] This study, no doubt, will be looked at with great interest and scrutiny, and more research on this topic is sure to follow. Perhaps NAC prevents damage to central vestibular circuits. It also seems possible that pharmaceutical countermeasures enhance the recovery of vestibular hair cells, just as

with auditory hair cells. Currently, there are no studies on this, but vestibular hair cells may be treated indirectly because there is also an attempt to treat hearing loss or TBI with medications.

TBI is frequently accompanied by headaches,[42,48] and they often are strikingly similar to primary migraines.[49–51] TBI is treated just as any condition with migraine-associated dizziness: by trying to decrease the number of acute headaches, which should decrease the associated dizziness. Treatment starts with a trial of diet and lifestyle modifications, followed by migraine prophylactic medications as needed. There is a tremendous variation in medication selection among providers, but it is important to select them carefully because they can impair cognition, reduce exercise tolerance, alter mood, and decrease temperature tolerance. Abortive migraine medications do not stop the dizziness, and these should be used in moderation to prevent rebound headaches.

Good doctors always take time to do the basics well, so be sure to do a Dix-Hallpike maneuver as long as the cervical spine is okay. Anyone dizzy after aural/head trauma may have benign paroxysmal positional vertigo (BPPV), even if the symptoms do not sound like BPPV. Many times, positive findings are indicated. Treating this condition makes an immediate improvement, although it is sometimes more difficult than with idiopathic BPPV.[52]

Duty limitations depend on the job, and many servicemen have jobs that require a high level of vestibular function. For example, consider walking on a trail with a weapon, carrying a load, and looking elsewhere. It makes good sense to progress into these sorts of activities by starting somewhere safe. As with hearing loss, the provider may need to talk to the patient's unit and work out a plan that enables recovery without jeopardizing anyone's well-being.

AURAL SOFT-TISSUE AND TEMPORAL BONE TRAUMA

The auricle and temporal bone can be injured in many ways in a combat environment, including from

- gunshots,
- fragments from IEDs and rocket-propelled grenades,
- falls (often after the IED or rocket-propelled grenade),
- vehicle crashes, and
- dog bites.

The result is often a mixture of insults, such as penetration, avulsion, contusion, and burn; but the basic principles of head and neck trauma still apply, whatever the etiology. The first thing to do is to verify that the primary trauma survey is complete and that more serious injuries are already addressed. In particular, an ear injury may be adjacent to an intracranial injury. This needs to be evaluated before worrying about the ear. It is also essential to assess and document facial nerve function as soon as possible. A wise otolaryngologist does not trust someone else's facial nerve examination.

Imaging is on a case-by-case basis and, to some degree, depends on what is available. Theater hospitals usually have CT (computerized tomography) scanners so that a noncontrast scan of the head is readily available for assessment of intracranial injury. These scans can be extended to identify fractures to the temporal bone, mandible, and cervical spine, depending on the situation. These additional images can be added with just a few more seconds of scanning, but it is important to communicate intentions with the trauma team at the time of the initial evaluation. However, there are times when the environment may be too urgent to do this, but it is best to ask and avoid a second trip to the scanner. If there is not a CT, plain X-ray films are still useful for the cervical spine and mandible, but rarely add useful information about the temporal bone. CT and plain X-ray films both work well to identify and locate foreign bodies, but note that even large fragments of wood, plastic, and glass may not be visible.

Be sure to thoroughly clean any wound without further damaging the tissue. One easy technique is to pierce the top of a 1-L saline bottle a few times with an 18-gauge needle and then use this to spray the wound. The liquid can also be tinted with povidone-iodine. Syringe irrigation with decent pressure works well, too. Motorized pulse lavage is a little rough and is not appropriate by the ear canal. Debris may have been blasted into the superficial dermis, and this can cause traumatic tattooing. Some of this debris can be removed with a surgical scrub brush, but be gentle or just use the sponge side. Chlorhexidine is ototoxic, so use povidone-iodine if there is a tympanic membrane perforation. Be very meticulous about removing foreign material (use loupes and a lot of patience) and only debride tissue that is obviously dead. Even tissue that appears nonviable sometimes survives on the ear.

Local anesthetic mixed with epinephrine is often used during debridement and repair because the bleeding is difficult to work through. However, epinephrine can be withheld if the auricle is partly detached. It is possible to save an ear even if nearly completely de-

tached. If reattachment fails, it can always be debrided later without harm, whereas the reconstruction of a new auricle is very challenging. There are even dramatic cases of entirely amputated ears surviving after reattachment, even without microvascular anastomosis.[53] Be careful not to wrap any dressings too tight. The ear might be better off with no dressing if any part of the auricle was reattached.

Suture preferences are often personal, but consider a follow-up plan before using a nonabsorbable suture. A local national may not get to see another doctor again. Sutures may be concealed by a scab and missed by subsequent providers if someone is transferred to another level of care. Nonabsorbable sutures are best used when the patient will remain under your care long enough to remove them. The provider can also communicate the sutures' presence directly with the next level of care, but this does not always work.

Antibiotics are probably not necessary for most auricular injuries unless dirty after debridement or if a large amount of cartilage is involved. This is a matter of opinion because there does not appear to be any studies that address this directly. Fluoroquinolones are a good choice because of their coverage of *Pseudomonas aeruginosa* and *Staphylococcus aureus*. Their oral bioavailability is also a distinct advantage. Topical antibiotics such as mupirocin or bacitracin make good sense when there is a lot of superficial damage or a burn. However, beware of increasing hypersensitivity to bacitracin.[54] There have been problems with this when it is applied to wounds indiscriminately.

Early closure of a periauricular wound is nearly always acceptable unless it looks infected, but reopen it immediately if signs of infection do develop. Absolutely beware of any deep wound closed by someone unknown, especially if that person is not a surgeon. The wound may look nice, but was probably closed without proper wound exploration. Some interesting objects have been found buried in these wounds, even with normal imaging. Now, almost routinely, open any fresh penetrating wound. Some restraint is necessary. There is no reason to dig around certain critical structures just to retrieve an object. For example, fragments have been found against the petrous carotid artery and in the transverse sinus. These were definitely best left alone or at least left for the experts to remove.

It is essential to take the time to piece all tissues back to where they belong. This is challenging, but surprisingly, bad-looking wounds can come together without tissue loss. It is best to avoid anything fancy if tissue is missing. A basic skin rotation or advancement flap is usually enough to fill a small defect around the ear. Otologists rely on three robust local-regional flaps for larger tissue defects: (1) the cervicofacial advancement flap, (2) the temporalis muscle flap, and (3) the superficial temporoparietal fascia flap. They are not hard to harvest and can be combined with skin grafts. Regarding the auricle, close any gap primarily (if possible) and save the flaps for later. There is no rush to reconstruct defects if the patient will be transferred soon to a higher level of care. Prepare the wound for the next surgeon by debriding it, piecing things together, and then covering the remaining defect with a moist dressing.

It is easy to overlook trauma to the ear canal. Clear out any debris and examine it. Lacerations or avulsions can be treated with a wick to prevent canal stenosis. More complex maneuvers (eg, skin grafts) are probably counterproductive.

If there is a severe injury to the ear canal, middle ear, or mastoid—especially if there is a large cerebrospinal fluid (CSF) leak—it may be necessary to obliterate and close the ear. This will leave the patient with a maximum conductive hearing loss, so it should not be performed for damage limited to superficial tissues. It also requires a microscope and a drill at a minimum, and there is no reason to perform this procedure on a patient who can be at a medical center within a few days. The basic procedure includes

- performance of a mastoidectomy;
- removal of the ear canal skin, tympanic membrane, malleus, and incus;
- occlusion of the Eustachian tube; and
- overclosure of the meatus (the auricle remains).

It is essential to remove all remnants of the ear canal and tympanic membrane or the implanted epithelium will grow into a cholesteatoma. The mastoidectomy does not have to be extensive and can be canal wall up if the ear canal is still intact. The mastoidectomy defect is filled with a temporalis muscle flap or abdominal fat. There are many ways to plug the Eustachian tube, but one easy technique is to trim the incus, put some bone wax on it, and firmly press it into the orifice. One advantage of obliteration is that it does not require much postoperative care compared with procedures that attempt to salvage a severely damaged ear canal. It works well for nearly any CSF leak, but smaller ones definitely can be repaired in less drastic ways.

Any CSF leak should be closed with the wound. Closing the superficial tissue may be enough for a small leak, but with anything larger around the ear, this will just divert the fluid through air cells and down the Eustachian tube. These leaks should be addressed where the dura is violated. Possible solutions include primary closure of the dura, or plugging with muscle,

fat, or fascia. The plugs sometimes can be sutured in place, and any repair can be reinforced with fibrin glue. The temporal lobe helps seal tegmen leaks, but leaks along the posterior fossa dura are a different matter. Large ones will require fat obliteration of a canal-wall-up cavity, or the previously described ear obliteration. As always, bedrest, head elevation, and stool softeners are important postoperative measures, but a lumbar drain is not suitable or available in the deployed setting.

An injury to the jugular bulb, sigmoid sinus, or transverse sinus can cause tremendous bleeding, as well as devastating neurological sequela. Fortunately, this is a low-pressure system, and elevating the head of the bed makes a big difference. If you suspect an injury, pack a large piece of oxidized cellulose gauze (eg, SURGICEL NU-KNIT, Ethicon, Somerville, NJ) into the wound and follow this with direct pressure. Typically, this is not the way to deal with a sigmoid sinus injury during mastoid surgery, but trauma is a far less controlled situation and exsanguination is the immediate concern. Leave part of the gauze outside of the sinus (or even suture it in place) to prevent it from entering the venous circulation as an embolus. Do not try to completely occlude the lumen of the sinus unless this is absolutely necessary to stop the bleeding. What if this is the only sigmoid sinus or what if there is no collateral venous flow through the posterior confluence of venous sinuses? Beware that exploring the opening can make it even larger. Once bleeding is controlled, either temporarily close the wound (this may prevent air embolism) or apply a pressure dressing. Then have the patient evacuated somewhere where a neurologist can assess the injury in a more controlled setting.

There is the possibility of caring for a local patient with a major injury who will never see anyone more skilled than the military provider who may perform a major reconstruction with less than ideal timing, training, or equipment. It is difficult to give any hard guidelines, but it is clearly important that the provider understand his/her own limitations. Trained microvascular surgeons have managed to do free flaps while deployed; but, for other physicians, it is far better to stick with the local-regional flaps previously described. There is a chance that a patient may be left with a less than satisfying reconstruction, but that is better than making things worse despite good intentions. Always remember that a more experienced colleague can be consulted for advice and encouragement.

SUMMARY

During deployment, the provider can expect to see a good deal of otological trauma. It is important to look for tympanic membrane perforations because simple measures may assist spontaneous healing and prevent cholesteatoma formation. Take acute hearing loss and tinnitus seriously because immediate treatments may have lifelong benefits. Therefore have a low threshold for providing otoprotective pharmacotherapy. Posttraumatic dizziness is complex. Most patients can be helped if the physician has a basic understanding of acute vestibular injuries, cervical dizziness, the postconcussive syndrome (mTBI), migraine-associated dizziness, and BPPV. Soft-tissue injuries can be challenging. They require patience and faithfulness to the basic principles of facial plastic and reconstructive surgeries. Temporal bone trauma requires close observance of intracranial, dural, vascular, and facial nerve injuries. Whatever the injury, do not overtreat someone who will soon be transferred to a higher level of care; instead, be sure that the patient is optimized for this transition. Remember, simpler is usually better.

A recurring issue in this chapter is that knowledge deficiencies can be clinically limiting. There is very little information on the pathophysiology and pharmacological treatment of auditory injuries caused by impulse noise or blasts. There is even less information on vestibular injuries. Thus, the following goals are necessary:

- better field diagnostics,
- basic science research to define the pathology of acute blast-induced and severe noise-induced hearing loss, and
- translational research to develop novel therapies for these injuries.

There is a lot of work in progress. It will take a long time to sort through the data available from Iraq and Afghanistan or to complete the basic science projects motivated by them. No doubt, some of this will be helpful, but it will not be enough. It is typical for attentiveness and financial support to diminish during peacetime, but these pursuits will need to be continued for whenever history decides to repeat itself.

Good care downrange requires more judgment than skill, although both are important. Still, it is more important to know when and when not to do something than to be technically gifted. It is impossible to anticipate every conceivable situation, but a positive attitude and flexibility from the outset will go a long way toward productivity and contentment.

CASE PRESENTATIONS

Case Study 20-1

Presentation

A contractor from Sri Lanka was taking a shower when a mortar came through the ceiling and exploded (January 2010). It injured the right side of his face and knocked him unconscious. He was intubated at the site and transported to the theater hospital in Balad, Iraq. A trauma surgeon and the head and neck team (including author PDL) evaluated him simultaneously. There was profuse bleeding from the wound despite direct pressure. Vessels within the wound were clamped, but there was still bleeding, so a neck exploration was done. The external carotid artery had an injury immediately distal to the bifurcation. This was controlled and ligated. It was also apparent that there was a major mandibular injury. Thus, after initial stabilization, a CT scan was performed.

Preoperative Workup/Radiology

CT showed a comminuted mandible fracture, but the temporal bone was uninjured (Figure 20-1). There was a parietal skull fracture, but no evidence of intracranial penetration or bleeding. The cervical spine appeared normal.

Operative Plan/Timing of Surgery

The team operated on this patient, but he was transported quickly out of Iraq and then back to Sri Lanka. Figure 20-2 shows him shortly after stabilization, although there was also tracheotomy for comfort and stability during transport. The function of the facial nerve was unknown. Over half of the auricle was missing, including all of the meatus, but the ear canal and tympanic membrane looked normal. The external wound also went into his oral cavity. It was decided not to repair the mandible fracture, but rather remove two molars and several loose fragments of bone, close the oral cavity laceration, and then let him swing. It was also determined that it would be easiest to amputate the rest of the auricle and then move in surrounding tissue to close the defect. So, the patient was taken back to the operating room immediately after the CT scan.

Operation

An otolaryngologist (author PDL) and an oral and maxillofacial surgeon performed the surgery. The wound was thoroughly irrigated and debrided, including removal of bone fragments and two molars. There were not any fragments. The oral cavity defect was closed primarily. Figure 20-3 shows the wound at this point, and the main trunk of the facial nerve is visible. No dissection was required to see the nerve like this. Further dissection showed that the entire nerve was intact. The auricle was amputated. The wound was closed using a cervicofacial rotation flap, a superficial musculoaponeurotic system flap, and undermining of the posterior scalp. The edges of these flaps were trimmed and approximated around the external auditory canal. This was held open with a wick. Figure 20-4 shows the patient 2 days after surgery.

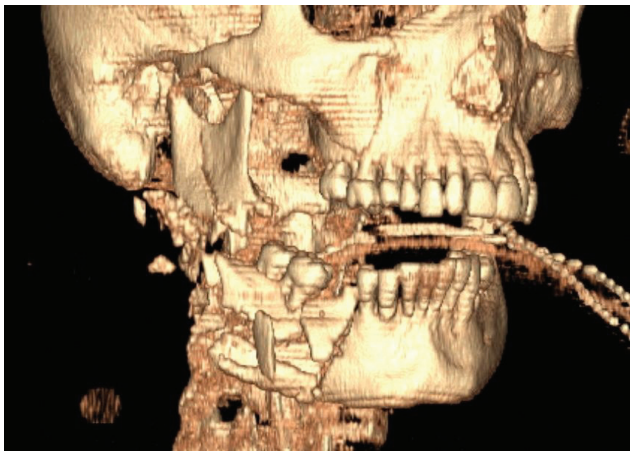

Figure 20-1. Three-dimensional reconstruction of CT (computerized tomography) scan.

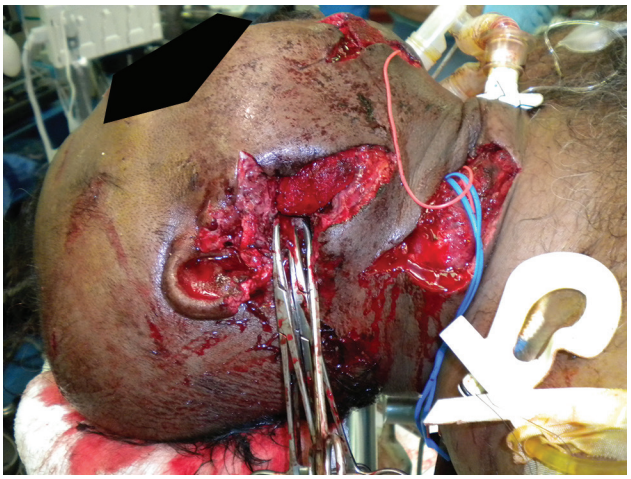

Figure 20-2. Mortar trauma following initial stabilization.

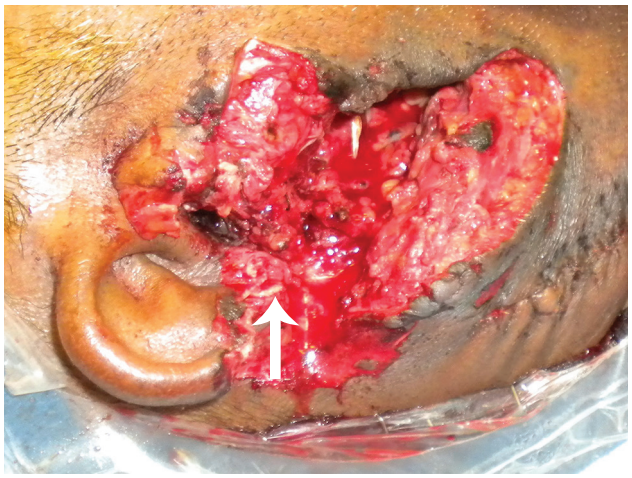

Figure 20-3. Partial auricular amputation. *Arrow* shows the main trunk of the facial nerve.

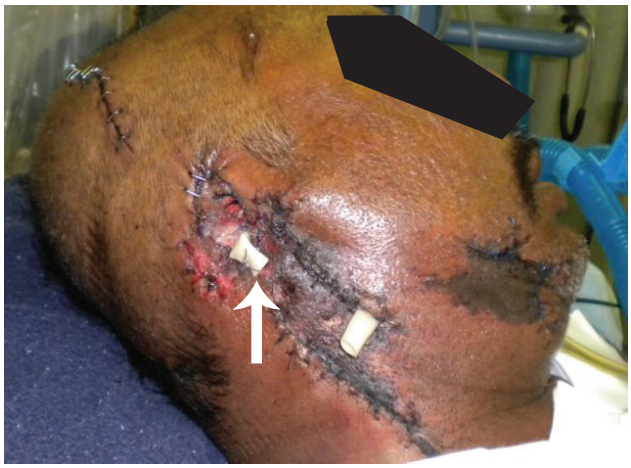

Figure 20-4. Following surgery. *Arrow* is on a wick in the external auditory canal.

Complications

None, but the patient was transported out after about 72 hours, and was lost to follow-up. He also required more than 20 units of blood.

Lessons Learned

This case would not have been treated any differently because it was understood from the outset that he would not have long follow-up with the team. It was vital to keep the reconstruction simple. There was one important exception. The patient wore eyeglasses, which was obviously difficult without an auricle. If the team had known that, then they could have preserved what was left of the auricle for the sake of the glasses, even though it would have been unsightly. The rationale was that an auricular prosthesis would look better and be easier to fit with a total amputation, but this probably was not a realistic possibility for him.

Case Study 20-2

Presentation

An adult Iraqi male was shot in the face by a sniper (January 2007). The bullet entered immediately lateral to the left lateral orbital rim and exited over the left mastoid (Figure 20-5). He reported severe left-side hearing loss, and a Weber examination lateralized to the right ear. He did not have vertigo or nystagmus. The left brow was paralyzed, but the facial nerve was otherwise normal. The ear canal skin and cartilage were lacerated, and there was blood behind an intact tympanic membrane.

Preoperative Workup/Radiology

A CT scan confirmed that the bullet fragmented on impact with the mastoid, leaving part of it embedded in the comminuted mastoid bone. The posterior canal wall was intact, but the superior ear canal was fractured. There were no intracranial findings, and the sigmoid sinus and the fallopian and carotid canals appeared normal.

Operative Plan/Timing of Surgery

It was decided to take this patient to the operating room as soon as possible to remove the fragment and repair the external auditory canal injury. There was no

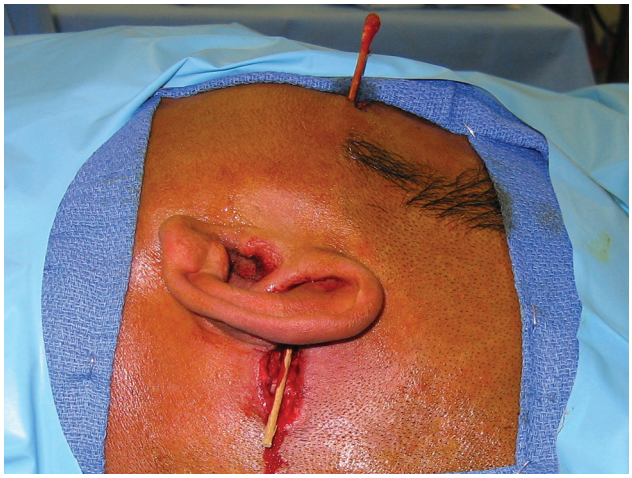

Figure 20-5. A cotton-tipped applicator inside a bullet wound.

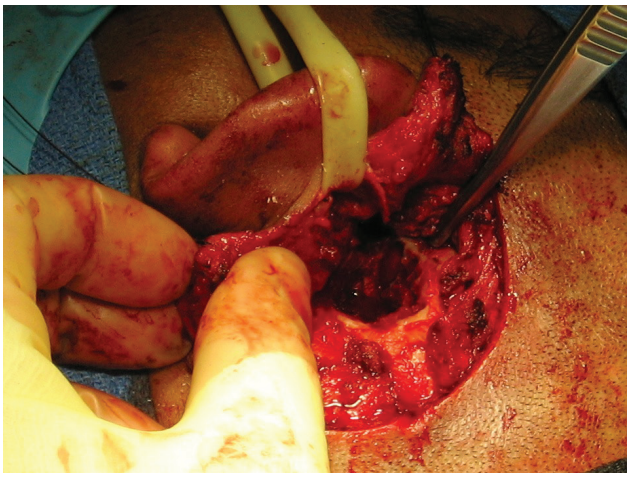

Figure 20-6. Intraoperative view of the limited mastoidectomy performed to remove bullet fragment.

otologic drill, but an orthopaedic drill was available. There was also a small microscope available from the clinic. There was no need to explore the facial nerve because only the temporal branch was paralyzed.

Operation

This case was treated by Colonel David Hayes. He used loupes and an orthopaedic drill with a small acorn-shaped cutting burr to remove comminuted mastoid cells, and to expose and remove the bullet fragment (Figure 20-6). He then switched to the microscope to enter the mastoid antrum. At that point, there was evidence of a slow CSF leak, but no obvious violation of the tegmen or posterior fossa dura. There were no visible injuries to the otic capsule. The mastoid segment of the facial nerve was not exposed, and the tympanic membrane was not elevated. A temporalis myofascial flap was rotated into the mastoid because of the CSF. The external auditory canal skin was reapproximated and then stented with a nonabsorbable sponge packing.

Complications

None immediately. The patient left the hospital 3 days after surgery and was lost to follow-up. His Weber examination was equivocal at the time of discharge.

Lessons Learned

This is a good example of using the equipment at hand to do what was necessary, which was to remove the fragment, repair the ear canal, and stop any CSF leak. It also shows good restraint. There was a need to expose the intratemporal facial nerve to accomplish these objectives. Although it is important to identify the facial nerve during most mastoid surgery, this would put it at a greater risk here because of limited visibility and an unfamiliar drill. This case also shows how a temporalis muscle flap can be used to stop a CSF leak, even if the site of the leak cannot be pinpointed. Unfortunately, it also shows how difficult follow-up can be and how difficult it is to learn from your own results.

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